# THE PATHOLOGY OF THE REPRODUCTIVE TRACT OF THE LAYING PULLET WITH NEWCASTLE DISEASE

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GANESWAR BISWAL

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R. UNIV. BIBLIOTHEEK LEIDEN

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# THE PATHOLOGY OF THE REPRODUCTIVE TRACT OF THE LAYING PULLET WITH NEWCASTLE DISEASE

Newcastle disease is known to cause a sudden and drastic drop in egg production (Beach, 1942; Berg, Bearse and Hamilton, 1947) and mortality of 0 to 5 per cent (Platt, 1948) in layers. The greatest loss among layers frequently results from reduced production and impaired egg-shell and albumen quality (Lorenz and Newlon, 1944; Berg *et al.*, 1947). Egg production reportedly returns to normal within 4 to 5 weeks (Platt, 1948). The pathogenesis of temporary cessation of egg-laying and of production of abnormal eggs has not yet been reported. This study of 73 laying pullets (48 White Plymouth Rocks experimentally infected, 6 White Leghorns naturally infected and collected at necropsy from 4 to 7 days after the initial symptoms and 19 White Plymouth Rock healthy controls) was made in an effort to shed light upon the subject.

Groups of 4 or 5 infected birds were sacrificed at 3, 5, 7, 9, 11, 14, 21, 28, 35 and 56 days after infection. Groups of 1 or 2 healthy controls were sacrificed at the same intervals. Ovarian and magnal tissues were collected for virus isolation. Tissues from ovary, infundibulum, magnum, isthmus, uterus, vagina and bursa of Fabricius (when present) were collected for histopathologic study. Serum-calcium and inorganic-phosphorus determinations were made before and after infection. Records were kept of weights of entire egg, yolk, albumen and shell, thickness of shell including membranes, and approximate pH of albumen.

The virus was isolated from the ovarian and magnal tissues of the birds from 5 to 7 days after infection. The infected birds went out of production for periods varying from 7 to 22 days. This temporary cessation in egg production was due to a marked degeneration of both mature and immature ovarian follicles accompanied by an inflammatory process. When this stage of degeneration was over, the birds came back into

production even though inflammatory changes were still present. The total yolk weight was not materially altered when the birds returned to production.

The infundibula of the infected birds showed only lymphocytic hyperplasia in the later stages of infection. In the magna pathologic changes varying from heterophilic infiltration, edema and focal necrosis to lymphocytic hyperplasia were observed. However, on the basis of total albumen weight, function of this section appeared to be normal. The isthmian glands of the naturally infected birds showed heterophilic infiltration. In the uterus the inflammatory changes varied from heterophilic infiltration, edema and capillary hemorrhage in early stages of the disease to lymphocytic hyperplasia, mild connective tissue proliferation and glandular atrophy in the later stages. The effect of the virus on the shell-secreting glands of the uterus lasted throughout the period of observation (56 days). This was evidenced by the fact that when the birds came back into production they showed decrease in shell weight and thickness in spite of normal serum-calcium levels. Some of the infected birds showed slight decreases in total egg weight. The pH of the albumen remained relatively constant.

While this study dealt primarily with pathology, a number of histologic features were observed which apparently have not been previously reported. In the ovarian stroma these included chromaffin cells, epoöphoron, sympathetic ganglion cells, hyaline bodies, plasma cells, extravascular erythrocytes and macrophages, and extrafollicular yolk spheres of varying size. Scattered lymphocytes were present in the cortex, medulla, ganglia, nerve trunk and in the interstices of the stroma of the ovary. The left adrenal gland seemed to be partially embedded in the ovarian tissue.

Scattered plasma cells, lymphocytes and lymphocytic aggregates were seen in all five segments of the oviduct of each healthy control bird. Parasympathetic ganglion cells were seen in the vagina only—in the poorly defined submucosa, between the two muscular layers, and in a subserous location or embedded in the longitudinal muscular layer near its peritoneal surface.

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## VITA

Ganeswar Biswal was born on January 2, 1919, in Golabandha, Angul, Orissa, India. After completing his primary education he entered the Government High English School, Angul, in 1929, as a government primary-school-scholarship holder. He passed his matriculation examination from the same High School under Patna University, Patna, India, in 1937. He joined the Bihar Veterinary College, Patna, as a government stipendiary and was graduated from said college with distinction in Veterinary Medicine and General Veterinary Surgery in 1940. From the time of graduation till 1948 he worked in different capacities in the Civil Veterinary Department, Orissa, India. In 1946 he underwent the Post Graduate Course Training at the then Imperial Veterinary Research Institute, Mukteswar, India. During the same year he was selected by the then Joint Public Service Commission of Bihar, Orissa and Central Province and Berar as a scholar for training broad. In 1948 he matriculated at Michigan State College, East Lansing, Michigan. In 1950 he received his D. V. M. (with honor) and M. S. degrees from the same college. Since 1950, he has been working as an assistant in the Department of Veterinary Pathology and Hygiene, College of Veterinary Medicine, University of Illinois.

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